Gangrene complicating dopamine therapy

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Dopamine is frequently used in the management of cardiogenic and septic shock because of its positive inotropic effects. Ischaemic changes in the extremities, such as bluish discolouration and coolness of the fingers and toes, may be observed with prolonged administration of the drug at high infusion rates[1]. However, progression of this ischaemia, resulting in gangrene, is uncommon.

CASE HISTORY

A man aged 40 was admitted with a 3-day history of high-grade fever with rigors and generalized bodyache. His temperature was 40 °C, pulse-rate 124/min and blood pressure 104/76 mmHg. Haemoglobin was 8.4 g/dL, blood urea 15 mmol/L, and serum creatinine 170 μmol/L; haemoglobinuria was present. Prothrombin time was 8 s longer than control, activated partial thromboplastin time was 13 s longer than control and fibrinogen degradation products measured 82 μg/mL. Disseminated intravascular coagulation (DIC) was diagnosed. A peripheral blood smear showed trophozoites of the malarial parasite *Plasmodium falciparum*. Intravenous fluids and chloroquine were administered. About 48 h later, oliguria developed. With optimum central venous pressure, systolic blood pressure was 96 mmHg and the urine output remained low. Therefore dopamine infusion was started at 8 $\mu g.kg^{-1}.min^{-1}$. A urine output of 25–30 mL/h was achieved. Eight hours later all the toes of both feet were observed to be cold and dusky. The dopamine infusion was immediately stopped; there was no substantial drop in urine output. Over the next 6 weeks, all the toes of both feet developed dry gangrene and required to be amputated.

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DISCUSSION

When dopamine is administered in low doses (2–5 $\mu g.kg^{-1}.min^{-1}$) it brings about vasodilatation in the renal and mesenteric vascular beds. In moderate doses (10–20 $\mu g.kg^{-1}.min^{-1}$), dopamine enhances cardiac contractility; higher doses (20–50 $\mu g.kg^{-1}.min^{-1}$) may also cause vasoconstriction[1]. Consequently, peripheral ischaemia and gangrene are not unexpected following the use of large doses of dopamine. However, in the patient reported here, the maximum dose of dopamine was only 8 $\mu g.kg^{-1}.min^{-1}$. Gangrene has only rarely been reported with dopamine infusion rates in the range of 1.5–10 $\mu g.kg^{-1}.min^{-1}$ [2–4].

Gangrene complicating low-dose dopamine therapy suggests either an idiosyncratic response to the drug or a multifactorial cause of the ischaemia and necrosis. Coexisting disseminated intravascular coagulation may be one risk factor[4], and the occurrence of gangrene in our patient, whose DIC was consequent to the intravascular haemolysis associated with malaria, adds to the evidence. Perhaps low-dose dopamine therapy, when superimposed on the hypercoaguable state of DIC, causes peripheral vessels to narrow below a critical diameter. Underlying atherosclerotic peripheral vascular disease, diabetic microangiopathy and hypotension might also contribute to the pathogenesis of the gangrene.

When dopamine is infused in a patient with DIC, we recommend close monitoring of the extremities for ischaemic changes. Once peripheral ischaemia is detected, the dopamine infusion should be stopped immediately. Benefits have been reported with intravenous infusion of chlorpromazine hydrochloride[5], infiltration of the ischaemic area with phentolamine hydrochloride[6] and local application of nitroglycerine ointment[7].

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